Objectif.– Comparer l'effet de la TB sur les pics de couple volontaire et réflexe (et les angles au pic) produits par le muscle rectus femoris (RF).

Méthode.– Étude pilote prospective ouverte. n = 15 patients blessés médullaires incomplets, gênés par une spasticité spécifique du RF (n = 20), avec hyperactivité en phase oscillante et Stiff-knee gait quantifié par une analyse quantifiée 3D de la marche (AQM), ont été évalués avant et après une injection de TB (Botox, 200 UI). Critères d'évaluation principaux : pic de couple (et angle au pic) du quadriceps, à différentes positions de hanche (0°, 90°) et vitesses angulaires lors de mouvements passifs (10, 90, et 150/s) et actifs (60/s) sur dynamomètre isocinétique. Les critères secondaires incluaient déficience (Échelle de Tardieu modifiée ; pic de flexion de genou et paramètres spatio-temporels en AQM), activité (Test des six minutes, Test chronométré de montée-descente d’escaliers), et gêne (ENS).

Résultats.– L’effet de la position articulaire (p < 0,01) sur le pic de couple réflexe confirma les hypothèses cliniques de sélection des patients. En post-injection, le pic de couple volontaire diminua (−16 %, p = 0,0004), sans diminution significative du pic de couple réflexe. En revanche, l’angle au pic réflexe augmenta (+5° à 90° ; p = 0,03), de façon concomitante à une amélioration clinique de la spasticité, du pic de flexion de genou en AQM (+4°, p = 0,01), des paramètres spatio-temporels, et du test de montée-descente d’escaliers (25 % ; p = 0,02).

Discussion.– Les auteurs ont discuté l’absence de diminution du pic spastique, ainsi que la pertinence clinique d’une faible amélioration analytique du pic de flexion de genou dans l’amélioration fonctionnelle apparente de la montée-descente d’escaliers.

Conclusion.– La TB a semblé retarder l’angle au pic du stretch-reflex, alors que le pic de couple volontaire a diminué. Après strict sélection, une injection de TB dans le muscle RF a abouti à une amélioration de la spasticité, de certains aspects fonctionnels de la marche, et de la gêne des patients.

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English version

CO20-001-e
Pathophysiologic and taxonomy in spastic paresis
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Deforming spastic paresis causes morphologic weakness (paresis), soft tissue shortening and muscle overactivity, including spasticity (increase in velocity-dependent reflexes to phasic stretch). Spasticity has long been considered as a major contributor to functional impairment in paretic patients. However, another form of muscle overactivity is today characterized in spastic paresis, antagonist cocontraction triggered by voluntary agonist command. Using electromyographic and dynamometric assessments during (i) isometric non-functional impact of antagonist cocontraction in spastic paresis, which may reverse the intended torque. Its occasional occurrence before the onset of agonist activity indicates supraspinal origin (misdirection of the supraspinal
drive at least in part. We define this stretch-sensitive, misdirected descending command as spastic cocontraction. While spastic cocontraction is really the main antagonistic mechanism impeding movement, in particular leg movements during gait, muscle overactivity is often assumed to be represented by mere measurements of resistance to passive movement. A five-step clinical assessment, including a quantitative assessment of active range of motion, may better serve the clinician to reflect the amount of cocontraction.

Another critical phenomenon is stretch-sensitive paresis, which is the aggravation of paresis of agonist command by the stretched position of the antagonist. The physiological uprinnings of spastic cocontraction and stretch-sensitive paresis probably involve neuronal rarefaction in agonist command, hypo-excitability of the remaining upper motor neurons, hyperexcitability of the lower motor neurons subserving the more shortened muscles and reduction or reversal into facilitation of Ia and groups II reciprocal inhibition.

Considering its determinants (agonist effort and muscle position), treatment of spastic cocontraction and stretch-sensitive paresis – and management of deforming spastic paresis in general – should logically involve a combination of:

– prolonged stretching postures for the more overactive and shortened antagonists, potentially in association with focal weakening agents such as botulinum toxin;
– intensive motor training (e.g. non assisted maximal amplitude rapid alternating movements and task-related exercises) of the less overactive muscles.

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Spasticity treatment by botulinum toxin in multiple sclerosis


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Keywords: Multiple sclerosis; Spasticity; Botulinum toxin

Botulinum toxin is now the reference treatment in focal spasticity, however, in the context of multiple sclerosis few studies have been conducted. The objective of this work is to try to clarify the use of botulinum toxin in multiple sclerosis, particularly in terms of indications.

Method.– We analyzed retrospectively all cases of patients with multiple sclerosis who received botulinum toxin injection on the pole Saint-Hélèr between 2007 and 2010.

Results.– During these 4 years, 673 patients including 126 people with MS have benefited from treatment with botulinum toxin. The population consists of 85 women and 41 men, mean age 49.4 ± 11 years with an average EDSS score of 5.8 ± 1.7 (2.5 to 9.5).

The most common indications are abnormal gait and difficulties in nursing. The lower limbs are usually involved, only 11 patients have benefited injection in the upper limbs. A specific indication was the vesico-sphincter dyssynergia of this work is to try to clarify the use of botulinum toxin in multiple sclerosis, however, in the context of multiple sclerosis few studies have been conducted. The objective of this work is to try to clarify the use of botulinum toxin in multiple sclerosis, particularly in terms of indications.

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Interest and tolerance of botulinum toxin for the treatment of spasticity in patients with lateral amyotrophic sclerosis

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Keywords: Lateral amyotrophic sclerosis; Spasticity; Botulinum toxin

Introduction.– Spasticity is a frequent and handicapping symptom in patients with LAS [1]. Before the use of botulinum toxin, the only treatment associated physiotherapy and oral medication.

Methods.– Forty-five patients suffering from LAS with either paraparesia (n = 10) or tetraparesia (n = 25) were referred to the specialized consultation in Physical Rehabilitation Medicine department in order to treat their spasticity. Some patients walked independently (n = 4), some required human or technical help for deambulation (n = 38), some were unable to walk (n = 3). Their medical charts were retrospectively analyzed, to determine the efficacy, tolerance and side-effects of botulinum toxin therapy.

Results.– Thirty-nine patients received botulinum toxin injections in their inferior members, in order to ameliorate ambulation (n = 36), sitting or nursing (n = 3). In 16 patients, a motor bloc of rectus femoris muscle was made. Five patients out of them were considered as ineligible for botulinum toxin therapy. Twenty-one patients were satisfied with the treatment that was continued. Eighteen patients stopped botulinum toxin therapy after the first injection either because of lack of improvement either by diseased progression. One patient experienced a transient respiratory degradation after treatment.

Discussion and conclusion.– Botulinum toxin therapy provided clinical amelioration of spasticity in more than a half of our patients. One patient had a transient respiratory degradation. To our knowledge, no study has been reported about the efficacy and tolerance of toxin therapy in spasticity management of LAS patients. This treatment is used to treat hypersialorrhoea, without severe side effect [2]. Nevertheless, the treatment should be started with low doses. As LAS patients often suffer from severe motor loss, motor bloc of the nerve of rectus femoris muscle should be perform before of botulinum toxin injections in this muscle.

References

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Evaluation of a treatment of focused spasticity with toxin botulinum

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Keywords: Focused spasticity; Clinical evaluation; Botulinum toxin; Vas satisfaction

Introduction.– Botulinum toxin is the first-line therapy in the management of focal spasticity.

Through this work we want to evaluate our modest experience with the use of this therapeutic method.

Materials and methods.– Fifty-two patients with focused spasticity due to various pathologies were treated with botulinum toxin in the year 2011. A pre therapeutic assessment and three weeks after injection of predetermined muscles was performed taking into account the following parameters:

– the pain intensity assessed by visual analog scale;
– spasticity measured by Ashworth scale;
– the range of motion measured by goniometry;